Reverse and Render and Opinion Filed May 8, 2018

In The
Court of Appeals
Fifth District of Texas at Dallas

No. 05-16-00609-CV

E.I. DU PONT DE NEMOURS AND COMPANY, Appellant
V.
VIRGIL HOOD AND LORRIE HOOD, Appellees

On Appeal from the 160th Judicial District Court
Dallas County, Texas
Trial Court Cause No. DC-13-03619

MEMORANDUM OPINION

Before Justices Bridges, Myers, and Schenck
Opinion by Justice Bridges

Appellees Virgil and Lorrie Hood (Hood) sued appellant E.I. DuPont de Nemours and Co. (DuPont) claiming Virgil's benzene exposure from DuPont's paint products caused him to develop acute myelogenous leukemia (AML). A jury found DuPont liable and awarded over $7 million in damages, including $1.5 million in punitive damages and $1 million in future medical expenses. The trial court granted DuPont's motion for judgment notwithstanding the verdict challenging the gross negligence finding and punitive damages award. The final judgment awarded Hood $6,985,535.25.

On appeal, DuPont argues the expert testimony presented at trial fails to establish causation and is therefore legally insufficient to support the verdict. It also challenges the trial court's refusal

to submit to the jury a design-defect liability question. Lastly, DuPont argues the evidence is insufficient to support the jury's award of future medical expenses.

Hood filed a cross-appeal challenging the trial court's order granting DuPont's JNOV and asks for reinstatement of the punitive damages award.

We reverse the trial court's judgment and render a take-nothing judgment in favor of DuPont.

Benzene and DuPont Paint Products

Benzene is an organic chemical compound present in crude oil. Through boiling, it can be distilled or refined for use in products such as paints.

It is accepted that benzene can cause some cancers such as AML. When a person breathes in benzene, it travels through the lungs, circulates to the bloodstream, and travels to the liver where it is metabolized. The benzene is metabolized by other cells within the bone marrow, which can affect the genetic material of the cell.

Other causes of AML include chemotherapy, radiation, and smoking. Regardless of these known causal links, in many cases, the cause of AML is unknown.

It is undisputed DuPont knew for decades that benzene, depending on the exposure and dose, could cause cancer. However, it is also undisputed DuPont never added benzene to any of its paint products or solvents. Rather, DuPont's paints and solvents were manufactured from xylene, toluene, naphtha, and mineral spirits, which all contain naturally-occurring amounts of benzene despite going through a distillation process. For example, toluene boils at one hundred ten degrees Celsius and is the hardest solvent to distill free of benzene, which boils at eighty degrees Celsius. Even distilling toluene twice will leave behind trace amounts of benzene. Xylene and naphthas distill at higher temperatures but even at the higher temperatures, benzene can get "caught up with some of these other molecules and comes off with them, instead of going all the

way up to the temperature at the top, where it should come off." As such, it is not feasible to distill every single solvent used in paint into a pure, benzene-free hydrocarbon. However, even though each of these hydrocarbons contains some trace amount of benzene, no regulatory body (such as the EPA, Agency for Toxic Substances and Disease Registry, or OSHA) or scientific body has concluded toluene, xylene, or naphthas is carcinogenic to humans.

In 1975, DuPont acknowledged "the toxicity of benzene is well publicized," and requested information regarding the amount of benzene in the solvents it purchased from its suppliers. DuPont wanted to establish a maximum benzene specification of 0.1% in its solvents.[1]

In 1978, DuPont verified that the benzene content in solvents purchased from its suppliers, with the exception of seven, met the 0.1% specification. DuPont encouraged the seven nonconforming suppliers to make acceptable products with a lower benzene content. During that time, DuPont also encouraged the purchase of products from only those suppliers who confirmed products at the benzene-specified concentration. However, some documentation indicated DuPont did not use its stated specifications "in full force."

Virgil Hood's Painting History

After graduating high school, Hood began working for Timpte, a tractor-trailer manufacturer. From 1973 to 1975, he worked as a painter. Hood recalled using mostly DuPont paint products and "seldomly" using Sherwin-Williams products.

Manufacturing of the tractor-trailers occurred as an assembly line process with the paint booth at the end of the line. Hood estimated the size of the paint booth was seventy feet long by twenty feet wide. Fans were located at one end of the booth to circulate air.

[1] A document from 1977, titled "DU PONT FABRIC & FINISHES DEPARTMENT RAW MATERIAL SPECIFICATION" lists benzene content as "0.1% Max."

Usually six to eight painters worked as a crew painting one tractor-trailer. An initial part of the painting process required use of a lacquer thinner to wipe down the tractor-trailer surface. After wiping down the surface, Hood sealed it with a DuPont primer. Next, he filled his paint gun with at least two quarts of paint and applied two coats to each trailer. He painted eight trailers a day (approximately one per hour). At the end of the day, he cleaned his paint gun using lacquer thinner. Paint was visible in the air and on his clothes.

Hood said it was mandatory to wear a respirator while painting, and although Timpte provided gloves, he did not wear them because they were flimsy latex that made it harder to paint. Timpte had "pretty strict" safety rules, and he knew it was very important to always wear the respirator. He signed a document on January 23, 1981 indicating he "received proper instruction on the use of the above personal safety protection," which included "respirator protection." He was fitted with a new respirator every six months or whenever he requested one. He never complained to Timpte about not having one.

Hood left Timpte in 1975 but returned in 1977. Upon his return, Timpte's facility had been "modernized" with a new paint booth, airless sprayers, and new fans. He continued painting until 1981.

Hood went to work for Continental Airlines as a painter in July 1984 and continued to work there for thirty years, except for a short time in 1994 when he was furloughed. He retired from Continental in December 2014.

In 2012, Hood began feeling tired and run down. After a physical, followed by blood tests and a bone marrow biopsy, he was diagnosed with AML.

Hood filed a products liability lawsuit against numerous defendants, including DuPont, Sherwin-Williams Co., and PPG Industries, Inc. He asserted that his exposure to solvents, primers, paints, lacquers, enamels, oils, petroleum products, and fuel designed, manufactured, sold, and

distributed by the defendants contained benzene, which caused him to develop AML. A jury found DuPont liable for defective design and manufacturing defects. It also found gross negligence. It awarded over $7 million in damages, including $1.5 million in punitive damages and $1 million in future medical expenses.

Du Pont filed numerous JNOV motions challenging the jury's findings. The trial court granted DuPont's JNOV motion challenging the jury's gross negligence finding and disregarded the $1.5 million punitive damages award. It also disregarded the jury's $60,000 award for past and future loss of household services. The trial court's final judgment awarded Hood $6,985,535.25. This appeal followed.

Expert Testimony Standard of Review

Throughout this case, both pre-trial and post-trial, DuPont challenged Hood's causation experts. DuPont continues to challenge the sufficiency of these experts' opinions to support the jury's verdict on appeal.

Under a traditional legal sufficiency standard of review, a reviewing court is to consider the evidence in the light most favorable to the verdict. *Bostic v. Georgia-Pacific Corp.*, 439 S.W.3d 332, 337 (Tex. 2014). The final test "must always be whether the evidence at trial would enable reasonable and fair-minded people to reach the verdict under review." *City of Keller v. Wilson*, 168 S.W.3d 802, 827 (Tex. 2005). However, the Texas Supreme Court has stated that when a sufficiency challenge involves the reliability and causation testimony of experts admitted at trial, "the reviewing court must undertake an almost *de novo*-like review and, like the trial court, look beyond the expert's bare testimony to determine the *reliability* of the theory underlying it." *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 710–20 (Tex. 1997); *see also Cooper Tire & Rubber Co. v. Mendez*, 204 S.W.3d 797, 804 (Tex. 2006) (appellate review encompasses the entire record, including contrary evidence tending to show the expert opinion is incompetent or

unreliable). To do otherwise would be to engage in a "meaningless exercise of looking to see only what words appear in the transcript of the testimony, not whether there is in fact some evidence." *Havner*, 953 S.W.2d at 712.

Expert testimony is unreliable if it is based on unreliable data or "if the expert draws conclusions from [his underlying] data based on flawed methodology." *Id*. at 714. "[I]f an expert's opinion is based on certain assumptions about the facts, we cannot disregard evidence showing those assumptions were unfounded." *Cooper Tire & Rubber Co.*, 204 S.W.3d at 804. Thus, when expert testimony is not grounded in the methods and procedures of science, it amounts to nothing more than subjective belief or unsupported speculation. *See E.I. du Pont de Nemours & Co. v. Robinson*, 923 S.W.2d 549, 557 (Tex. 1995). In this context, if the expert's testimony is unreliable, it is no evidence of causation and will not survive a sufficiency challenge. *Havner*, 953 S.W.2d at 713.

General and Specific Causation in Exposure Cases

In *Havner*, the Texas Supreme Court discussed a plaintiff's burden to prove both general and specific causation. General causation exists when a substance is capable of causing a particular injury or condition in the general population. *Id*. Because direct experimentation may not be possible to prove causation, a plaintiff may try to demonstrate that exposure to the substance at issue increases the risk of the particular disease through epidemiological studies. *Id*. at 715. Such studies examine existing populations to attempt to determine if there is an association between a disease or condition and a factor suspected of causing the disease or condition. *Id*. The supreme court held "epidemiological studies showing that the population exposed to a toxin faced more than double the risk of injury facing the unexposed or general population could be used to establish causation." *Id*. at 708; *Bostic*, 439 S.W.3d at 347. However, this is not a "litmus test" or "bright-line boundary," and a single study will not suffice to establish legal causation. *Havner*, 953 S.W.2d

at 718 ("[W]hen a number of studies have been done, it would not be good practice to pick out one to support a conclusion."); *Bostic*, 439 S.W.3d at 347.

When considering epidemiological studies, courts are also instructed to consider the "significance level" or confidence level of the studies. *Havner*, 953 S.W.2d at 723–24; *Bostic*, 439 S.W.3d at 347. To be considered scientifically reliable, an epidemiological study must (1) have a relative risk of 2.0 and (2) be statistically significant at the 95% confidence level. *Havner*, 953 S.W.2d at 723.[2] An expert "cannot dissect a study, picking and choosing data, or 'reanalyze' the data to derive a higher relative risk if this process does not comport with sound scientific methodology." *Id*. at 720. The *Havner* court emphasized that "even if a statistically significant association is found, that association does not equate to causation." *Id*. at 724.

However, general causation is never the ultimate issue of causation tried to the finder of fact in exposure cases. *See Bostic*, 439 S.W.3d at 351. General causation as established through epidemiological studies is relevant only insofar as it informs specific causation. *Id*. "Where direct evidence of specific causation is unavailable, specific causation may be established through an alternative two-step process whereby the plaintiff establishes general causation through reliable studies, and then demonstrates that his circumstances are similar to the subjects of the studies." *Id*. (discussing *Havner*). This burden includes proof that (1) the injured person was exposed to the same substance, (2) the exposure or dose levels were comparable to or greater than those in the

[2] By way of example, the *Havner* court explained the concept as follows:

> If, based on a confidence level of 95%, a study showed a relative risk of 2.3 and had a confidence interval of 1.3 to 3.8, we would say that, if the study were repeated, it would produce a relative risk between 1.3 and 3.8 in 95% of the repetitions. However, if the interval includes the number 1.0, the study is not statistically significant or, said another way, is inconclusive. This is because the confidence interval includes relative risk values that are both less than and greater than the null hypothesis (1.0), leaving the researcher with results that suggest both that the null hypothesis should be accepted and that it should be rejected.
>
> [A] study may produce a relative risk of 2.3, meaning the risk is 2.3 times greater based on the data, but at a confidence level of 95%, the confidence interval has boundaries of 0.8 and 3.2 The results are therefore insignificant at the 95% level.

Id. at 723.

studies, (3) the exposure occurred before the onset of injury, and (4) the timing of the onset of injury was consistent with that experienced by those in the study. *Id*. In *Bostic*, the supreme court emphasized "proof of substantial factor causation requires some quantification of the dose resulting from [plaintiff's] exposure to [defendant's] products." *Id*. at 355 ("dose matters"). However, the court conceded those in the studies need not exactly match the plaintiff's exposure, but "the conditions of the study should be substantially similar to the claimant's circumstances." *Id*. at 359.

With these concepts in mind, we consider whether Hood's two experts, Dr. James Stewart and Dr. Sheila Butler, provided reliable and therefore legally sufficient evidence to establish a causal link between Hood's benzene exposure through his use of DuPont's paint products and his AML diagnosis.[3]

Dr. James Stewart

Stewart, an industrial hygienist, calculated Hood's benzene exposure thereby determining his "dose." An industrial hygienist anticipates, recognizes, evaluates, and controls occupational health problems. Stewart has worked in the field of exposure assessment and exposure reconstruction for over thirty years. He described reconstruction as going back in time and determining the exposures that occurred. Exposure assessment is the process of looking at the individual person. Occupational exposure is the quantity of the relevant substance a person is exposed to in an eight-hour day.

Stewart calculated Hood's dose of lifetime benzene exposure using a computer model called ART.[4] The model, which was released in 2010 after five years of development, is designed to screen for potentially hazardous exposures across entire populations as part of the European

[3] DuPont has not challenged the qualifications of either expert witness.

[4] ART is an acronym for "advanced reach tool." Reach is an acronym for "registration evaluation and authorization of chemicals."

regulation of chemicals. It was developed by industry, academic and government experts, and has been peer-reviewed. It predicts a range of possible exposures based on a series of drop-down menus. The user does not enter data into the program, but instead chooses the closest option within the categories provided in the drop-down menus.

After Stewart selected certain data options for ART, the model calculated the dose of Hood's lifetime benzene exposure. Stewart testified Hood's total cumulative lifetime benzene dose from DuPont's products ranged between 8 and 49 parts per million years, with the mean and most likely average being 28.98 parts per million years.[5]

DuPont filed pretrial motions arguing ART was unreliable as a scientific tool to calculate exposure and challenging Stewart's underlying methodology in computing a reliable benzene exposure dose. The trial court overruled DuPont's objections and allowed Stewart to testify. At the conclusion of trial, DuPont moved for a directed verdict based, in part, on insufficient evidence of causation, which the trial court denied. DuPont continued to challenge the sufficiency of Stewart's opinions in its motion for JNOV.

On appeal, DuPont challenges both the general scientific reliability of ART and the data Stewart used to compute Hood's lifetime benzene exposure dose. Because we conclude Stewart's ultimate opinion is unreliable for the reasons explained below, we assume without deciding ART is accepted in the scientific community as a tool to calculate lifetime benzene exposure. Accordingly, we focus our analysis on explaining why certain data Stewart used to calculate

[5] Stewart explained parts per million years by way of example:

> So a railroad car - - railroad car, those big tankers, the black tankers that go down the railroad tracks, if you fill it with Ping-Pong balls, you got a million Ping-Pong balls in there, there's going to be a red one, right? That's one part per million . . . .
>
> So if I'm a painter and I'm exposed to one part per million for one year, I get one part per million year, right. If I paint for five years at one part per million, I would got [sic] five PPM years, parts per million years. All you're really doing is multiplying the concentration times the years that you did it. And that's the measure that's useful in benzene litigation or actually in the scientific literature also.

Hood's lifetime benzene exposure dose is unreliable, and therefore, amounts to no evidence of causation.

To support its sufficiency challenge, DuPont contends Stewart incorrectly inputted the following information into ART: (1) Hood's respirator use; (2) the amount of benzene in DuPont products; (3) spray application rates; (4) painting room size; (5) size of the vehicles being painted; (6) exposure to benzene from DuPont's products while using other manufacturers' products; (7) benzene exposure when Hood was not painting; and (8) air ventilation in the paint room.

Hood argues DuPont failed to preserve some of its challenges to the input data by not objecting to the trial court. DuPont responds an objection was not required to preserve these issues.

A review of the record, including DuPont's pretrial motions to strike Stewart's expert testimony, a hearing on the motion to strike, and its post-judgment motions challenging the sufficiency of the evidence on causation, reveals that DuPont challenged the reliability of the inputted data for (1) respirator use; (2) the amount of benzene in DuPont's products; (3) benzene exposure when Hood was not painting; and (4) size of the vehicles being painted. Thus, we consider whether Stewart's lifetime benzene exposure dose was unreliable based on the specific data inputted in ART for these categories. *See Havner*, 953 S.W.2d at 714 (noting expert's opinion unreliable if based on unreliable data).

1. Benzene Content of DuPont's Products[6]

Stewart testified benzene concentration in a product is the "main variable" when assessing exposure. DuPont argues Stewart assumed DuPont's products always contained exactly 0.1 to 0.5 percent benzene, which is contrary to the evidence. Further, it contends Stewart chose a range to fit ART's drop-down menus, which required him to choose between "extremely small" (benzene

[6] Stewart testified he calculated Hood's lifetime benzene exposure dose based on his time at Timpte from 1973-75 and 1977-81 because this is when his exposure would have been the greatest. Although Hood worked longer at Continental, Stewart explained Hood had a "mix of duties" including jobs other than painting.

content 0.1–0.5 percent or 1,000-5,000 ppm) and "minute" (benzene content below 0.1 percent or 1,000 ppm). Stewart chose the "extremely small" range based on "five or six other documents" in approximately thirty thousand DuPont documents he reviewed.

Numerous documents from manufacturers were admitted at trial regarding the amount of benzene in the hydrocarbons supplied to DuPont. While many of these indicated the manufacturers were in fact meeting DuPont's less than 0.1% expectation prior to its 1978 request, some were not:

- October 28, 1976 letter from Charter Chemicals indicating toluene and n-Hexane contained .2% benzene.[7]

- November 18, 1976 letter from Tenneco Oil indicating it may have supplied toluene with a benzene content between 0.03–0.1%.

- January 28, 1977 letter from Shell Chemical Company indicating one solvent (Tolu Sol 10) contained 0.2% benzene. Shell recognized "[f]rom time to time these values may change due to crude slates for refinery intakes. However these values are as good as we have at the present time."[8]

- July 5, 1977 letter from Exxon Company, U.S.A. stating that all of the products have "never exceeded 0.05% benzene." However, at that time Exxon did not routinely analyze for benzene so it had "reluctance" to guarantee results "for tests that we do not run consistently."

- April 11, 1978 letter from Chevron USA regarding benzene content of Chevron thinners stating "content of Chevron thinner 250 typically between 0.2 and 0.5 pct."

Further, Claiborne Smith, DuPont's environmental and training manager, testified before OSHA in 1978 regarding the agency's proposed 0.1% exemption to the benzene standard in products. He encouraged OSHA to adopt a benzene standard in favor of a 1% (or 10,000 ppm) exemption. His testimony indicated DuPont's main concern was the cost to DuPont of relabeling and pulling products from the shelves that admittedly violated the proposed lower exemption:

[7] A second letter from Charter, dated April 10, 1978, indicated all its products contained 0.1% or less benzene except for toluene which continued to be above DuPont's requested minimum at 0.12%.

[8] This indicated Shell could not always guarantee what was in its product because it depended on the amount of benzene in the crude oil refined by the refinery.

> As I've already stated, due to impurities in organic solvents, levels of .1 to 1 percent benzene are present in a large number of products which have already been packaged. These products are now in warehouses and the customer plants. In many cases, it would be physically impossible to locate them. Even if location was possible, the cost of locating and relabeling them and performing the initial monitoring would be exorbitant. We estimate the cost to du Pont and its customers would be $31 million.

Stewart also relied on a 2007 statement from an Exxon doctor admitting benzene levels in its products had been below .1% for approximately twenty years, or since 1987.

Stewart acknowledged a DuPont document indicating thirty-seven of thirty-nine solvents contained less than .1% but he questioned the data because "all they have to do is tell somebody that it's .1 or less. . . . there is no analytical method specified."

Stewart's testimony, along with manufacturers' letters confirming benzene levels of certain solvents, provided evidence that DuPont paint products contained levels of benzene within the ART input range chosen by Stewart during the time Hood worked at Timpte. Stewart explained why he relied on documentation showing DuPont products contained benzene levels higher than .1% and why he did not believe contrary documentation was true. Accordingly, Stewart did not rely on assumed facts that varied from actual facts. Rather, he determined based on his expert opinion which data was most reliable and used that inputted data for ART. As he stated, "[I]t's just not realistic to think you're going to buy from different suppliers and get the same exact number. And, ART requires you to select that range because the model was developed with that range in mind." Thus, his opinion was not contrary to actual, *undisputed* facts. *See Caffe Ribs, Inc. v. State*, 487 S.W.3d 137, 144 (Tex. 2016). As such, Stewart's dose exposure opinion is not unreliable because he chose the "extremely small" option in the ART model to calculate Hood's lifetime benzene exposure.

2. Respirator Use

Stewart testified the safety program implemented by a company and a painter's use of a respirator are both important considerations in conducting an exposure reconstruction. DuPont argues Stewart assumed that Hood's respirator failed most of the time and that DuPont had a poor safety program. Hood responds that factual conflicts regarding data input do not invalidate an opinion but instead fall within the province of the jury to resolve.

According to OSHA standards, a respirator should provide a ten times protection factor, meaning that if Hood used a respirator correctly, the air inside the mask would be one-tenth of whatever was outside. Although Timpte's respirator had a ten rating, Stewart opined it did not work at that level. This opinion was based on deposition testimony from Hood in which he said he got dizzy while cleaning with lacquer thinner. Stewart, however, never talked to Hood. Stewart could not testify as to how often Hood smelled vapors or got dizzy, and ART did not allow entry of such information.

Instead of trying to determine the effectiveness of Timpte's respirator based on Hood's generalized statements of smelling vapors "often" and "once in awhile," Stewart adjusted the impact factor of respirator effectiveness based on an unidentified 1972 peer-reviewed study. The study involved testing the effectiveness of workers' respirators within a workplace known to have "a lousy program" that was poorly implemented.[9] The results provided data on the protection factor range for a poorly implemented respirator program. Stewart testified "the low was like 1.2, I think, yeah, up to 11." Based on this data, he used the whole range for the protection factor of Hood's respirator, which he described as "the high and the low and all of the values in between." Although he never assumed Hood's respirator did not work at all, he admitted ART "assumed that it worked poorly or well and anything in between." Stewart ran 50 to 60,000 different computer

[9] He acknowledged such a study would not be approved today.

simulations in which he inputted a protection factor based on respirator effectiveness from 1.2 to 11.2.

The record provides little other information about this 1972 study. In establishing specific causation, the supreme court explained that while the studies relied on need not exactly match the plaintiff's exposure when establishing specific causation, "the conditions of the study should be substantially similar to the claimant's circumstances." *Bostic*, 439 S.W.3d at 359. Hood has not cited this Court to the 1972 study, and it does not appear to be in the voluminous record. As such, the record is devoid of any information about this study, except the year it occurred and that it involved a poorly implemented program.[10] Without information such as which type of industry was involved, which hazardous chemical was measured, the type of facility and ventilation, and the type of respirators used, it was improper for Stewart to rely on this study and then assume the protection factor related to Hood when he worked at Timpte. Stewart's testimony that the numbers were based on "good science" and not manufactured is conclusory and unreliable.

Hood seems to imply Stewart justifiably relied on this 1972 study because Timpte had a poor respiratory program and that alone was enough to use as a comparison. However, even assuming Timpte had a poor respiratory program, Stewart did not explain how the conditions of the study are substantially similar to Hood's working conditions. *See id*. ("conditions of the study should be substantially similar to the claimant's circumstances").

Moreover, Stewart assumed Hood's respirator was not working properly because he became dizzy "once in awhile" when climbing a thirteen-foot ladder to clean trailers with a lacquer

[10] Stewart refers to this study in his expert report and states the following:

> In 1998 OSHA published data from a study in 1972 where workers in poorly implemented respiratory protection programs were studied to assess the workplace protection factors (WPF), i.e., the level of protection actually provided in the workplace. The results were that WPFs ranged from 1.2 to 11.9 for these workers. To account for the poor respiratory protection program at Timpte, this range (as a uniform distribution) of WPFs was used to account for the use of respirators by Mr. Hood at Timpte.

He then cites to "OSHA, Preamble to the Final Rule, *Respiratory Protection*, Section V, Table V-1, January 8, 1998."

thinner. Stewart did not determine why the respirator was not working properly except to speculate it could have leaked because it did not fit properly while Hood was on a ladder. Dr. Stewart never talked with Hood to verify these assumptions.

Further, the wide range of Stewart's protection factor likewise assumed Hood's respirator failed more often than it worked—meaning the respirator provided only 1.2-fold protection (almost none) the same proportion of the time it provided 11.2-fold protection or any other protection value in between. Thus, he determined the respirator failed to meet OSHA's minimum rating of 10 most of the time, even though OSHA determined the respirator would provide ten-fold protection 95% of the time.[11] Built into this assumption, was yet another assumption—specifically, that Hood's respirator failed when he performed other tasks, like painting. No evidence supports this assumption. Hood said he smelled fumes and experienced dizziness when he wiped the trailers with lacquer thinner.[12] He did not say this occurred while painting. Thus, to the extent Hood contends Stewart's respirator adjustment "gave DuPont the benefit of the doubt," we disagree. Stewart's inferences are based on nothing more than Hood's testimony that he experienced occasional dizziness. When the facts support several possible conclusions, only some of which support the expert's conclusions, the expert must explain to the factfinder why those conclusions are superior based on verifiable evidence, not simply the expert's opinion. *Houston Unlimited, Inc. Metal Processing v. Mel Acres Ranch*, 443 S.W.3d 820, 832 (Tex. 2014). Stewart failed to explain why his conclusions were superior to other, equally plausible inferences such as the respirator cartridge needed to be replaced.

Stewart also justified his opinion that Hood's respirator did not provide ten-fold protection 95% of the time because that level of protection only applies when a respirator is properly used

[11] Stewart agreed that the model predicted that while Hood worked at Timpte, if his respirator worked, the level of benzene that he would be exposed to was below OSHA's occupational health safety standard of one ppm (.979).

[12] Hood spent approximately 100 minutes a day wiping down a trailer in preparation for painting.

based on an appropriate training program. Stewart assumed Timpte had a poor training program because of past OSHA violations. He also assumed the document Hood signed acknowledging he was properly trained was nothing more than a "CYA" form employees often felt obligated to sign.

OSHA citations for regulatory violations in 1984 (several years after Hood left Timpte) cannot support Stewart's conclusion the respirator program was poor. Violations from a one-time visit, none of which involved the painting area where Hood worked, do not support Stewart's assumptions that Timpte provided faulty respirators years earlier.[13] Moreover, Stewart admitted he discovered the violations after giving both depositions and forming his opinion. Although an expert may modify his opinion based on refinements to calculations or expand an opinion based on subjects already disclosed through the time of trial without invoking a need to supplement, Stewart's new testimony was neither. *See Vela v. Wagner & Brown, Ltd.*, 203 S.W.3d 37, 53 (Tex. App.—San Antonio 2006, no pet.) His pretrial reports stated he believed Timpte's protection program was inadequate because Hood experienced dizziness while wearing the respirator. His opinion was not based, in part, on OSHA violations and his use of them after the fact does not support his opinion.

To the extent Stewart assumed Timpte employees signed a "CYA" form without any real training, his assumption is unfounded. Stephen Silvas worked as a Timpte production control manager, plant manager, and vice president of manufacturing during the time Hood was employed. He testified the union worked closely with the plant on safety issues and conducted internal safety audits because OSHA conducted surprise audits. He said safety meetings were always happening based on any identification of an issue that needed to be discussed. As part of Timpte's respirator program, employees received ongoing safety talks and reviews of their respirators.

[13] The violations involved ozone within the foam department and not benzene within the paint department.

On January 23, 1981, Hood signed the "CYA" document indicating he had "received proper instruction on the use of the above personal safety protection," which included "respirator protection." Hood testified it was mandatory to wear a respirator while painting. He testified Timpte had "pretty strict" safety rules, and he knew it was very important to always wear the respirator. Hood further described the rules and policies as "quite drastic, you know, quite intense." If he needed protective equipment, he could request it. He was fit with a new respirator every six months or whenever he requested one.

Stewart provided no evidence, other than his conclusory belief, that the Timpte document was simply a "CYA" form. Rather, Hood testified the rules were "strict," and he knew the importance of wearing a respirator. Thus, the evidence indicates Stewart's opinion about the adequacy of Timpte's safety program was based on unfounded assumptions. *See Cooper Tire & Rubber Co.*, 204 S.W.3d at 804 (noting we cannot disregard evidence showing expert's assumptions were unfounded).

Because Stewart relied on a dissimilar study to determine the protection factor of Hood's respirator rather than attempting to compute the protection factor based on similar conditions at Timpte at the time of Hood's employment and because he made unfounded assumptions about the adequacy of Hood's respirator and Timpte's respirator program, we conclude Dr. Stewart's inputted data for the protection factor is unreliable.

3. Adjustment for Exposure Based on Non-Painting Job and Absenteeism

DuPont also challenges Stewart's exposure analysis based on inaccurate estimations for how many days Hood worked as a painter at Timpte. Specifically, DuPont argues Stewart failed to account for the months Hood worked as a forklift driver and was absent from work.

The length of time performing a task impacts exposure. In fact, Stewart "absolutely" agreed it was important to know how long a worker was exposed to a hazardous substance.

Stewart testified he subtracted six months' worth of days for the time Hood worked as a Timpte forklift driver. Documentation shows Hood became a forklift driver effective September 29, 1980. Stewart acknowledged Timpte was a "union shop" where job titles were strictly observed. Stewart agreed Hood left Timpte on November 18, 1981, meaning Hood was not exposed to DuPont products for the last thirteen months of his employment, yet Stewart only subtracted six months of days from his exposure analysis. Stewart testified that although a personnel document stated when the job change became effective, "It doesn't say how long it lasted. He said how long it lasted." The record does not expound on who "he" is but it most likely refers to Hood, who testified in his deposition that he spent "maybe six months" as a forklift operator. However, when asked at trial about his time as a forklift driver, Hood testified as follows:

> Q. And that was about - - according to your memory, about a six-month period of time that you were operating a forklift?
>
> A. Yeah.
>
> Q. Could it have been longer than that?
>
> A. It could have been. You know, I'm not sure exactly. I can't give you exact numbers.

The record indicates Hood's recollection about his time as a forklift driver is imprecise, but DuPont produced documentation from Timpte establishing he changed positions and worked as something other than a painter for thirteen months before quitting. These documents, which were not challenged, indicate how long Hood worked as a forklift driver and we may not disregard them. *See Cooper Tire & Rubber Co.*, 204 S.W.3d at 804 (noting we cannot disregard evidence showing expert's assumptions were unfounded).

Even if we accepted Stewart's conclusion that Hood only worked for six months as a forklift driver, it is undisputed Hood continued to work at Timpte, in some capacity, for another seven months. Nothing in the record indicates Hood then returned to a position in which he was

exposed to benzene. Accordingly, we conclude Stewart's inputted data for number of days exposed to benzene while working at Timpte is unreliable because he used inaccurate data to calculate the length of time Hood was exposed to benzene. *See Havner*, 953 S.W.2d at 714 (expert testimony unreliable if based on unreliable data).

In reaching this conclusion, we need not address whether Stewart's inputted data is further flawed for failing to consider days Hood was absent from work. *See* TEX. R. APP. P. 47.1. Likewise, having concluded some of the inputted data Stewart used in ART to determine Hood's lifetime benzene exposure resulted in an unreliable dose, we need not consider whether the dose was unreliable because Stewart made certain assumptions about spray painting large vehicles, which were not fully analogous to DuPont's working conditions. *See id*.

4. Conclusion

This is not a case in which the dose expert failed to offer any basis for his opinion. But our review requires this Court to look beyond the expert's statements. *Havner*, 953 S.W.2d at 713 ("The underlying data should be independently evaluated in determining if the opinion itself is reliable."). Stewart identified the factual basis on which he relied to calculate benzene exposure, but the facts on which he relied to make the calculation do not actually support his opinion, and each material part of an expert's theory must be reliable. *Whirlpool Corp. v. Camacho*, 298 S.W.3d 631, 637 (Tex. 2009). "It is incumbent on an expert to connect the data relied on with his or her opinion and to show how that data is valid support for the opinion reached." *Id*. at 642; *Mel Acres Ranch*, 443 S.W.3d at 831. Dr. Stewart failed to connect the data to his opinion by using reliable, undisputed facts. Instead, he relied on assumptions and then simply picked the median exposure number from the range ART calculated, which meant fifty percent of the predicted exposures would be lower than the ART results Stewart relied on at trial. Stewart admitted an expert could select any value within this range, even the extremes. We recognize Stewart was not required to

calculate a dose with mathematical precision. *See Borg-Warner Corp. v. Flores*, 232 S.W.3d at 765, 773 (Tex. 2007). However, we cannot conclude picking the median of a sweeping range of possibilities already plagued by assumptions is scientifically reliable to establish causation. *See, e.g., Castellow v. Chevron USA*, 97 F. Supp.2d 780, 792 (S.D. Tex. 2000) (when expert modeling approach is extremely sensitive to fluctuations in data and based on invalid or non-existent data, then "there is no hope that [expert's] technique, much less his results, are going to be reliable"). As the supreme court emphasized in *Bostic*, "dose matters," and such result-driven methodology is rife with error and speculation. As such, Stewart's calculation of Hood's lifetime exposure dose of benzene is unreliable and therefore no evidence supporting causation.[14] We sustain DuPont's second issue.

Dr. Sheila Butler

Butler, using the dose provided by Stewart, compared epidemiological studies to supply the alleged causal link between Hood's lifetime cumulative benzene dose and his development of AML. Because Stewart provided an unreliable dose, which Butler relied on and concluded was a substantial factor in causing Hood's AML, her testimony is likewise no evidence of specific causation.[15] *See Havner*, 953 S.W.2d at 714 (if expert relies on unreliable foundational data, any opinion drawn from the data is likewise unreliable); *see also Gharda USA, Inc. v. Control Solutions, Inc.*, 464 S.W.3d 338, 352 (Tex. 2015) ("interrelated expert testimony cannot be used to form a hybrid for which no [single] expert can offer support"). However, even assuming

[14] Hood cites two cases in which courts have admitted Stewart's expert opinion on exposure estimates. In *Milward v. Acuity Specialty Products Group, Inc.*, Rust-Oleum Corporation challenged the expert opinions of both Stewart and Butler in which they testified as a team to establish causation of Milward's APL from benzene in Rust-Oleum products. 969 F.Supp.2d 101 (D.Mass. 2013). The district court concluded Stewart's exposure estimate using ART was reliable; however, it excluded Butler's causation opinion. *Id*. at 108, 115. On appeal, the First Circuit affirmed the district court's ruling excluding Butler's testimony. Based on its disposition, it did not reach Rust-Oleum's challenge to Stewart's opinion. 820 F.3d 469, 471 n.1. In the second opinion, Stewart did not use ART to calculate benzene exposure. *See Schultz v. Azko Nobel Paints, LLC*, 721 F.3d 426 (7th Cir. 2013), *on remand*, *Schultz v. Glidden Co*., No. 08-c-919, 2013 WL 4959007 (E.D. Wisc. Sept. 9, 2013).

[15] Butler testified she relied on Stewart's industrial hygiene report to reach her opinion that benzene-containing paint products contributed to Hood's AML. She explained, "It would be impossible for me to determine just from an interview or reading a medical record how much of that product was benzene and how much of that benzene got into - - potentially got into his system. . . . I would rely on an industrial hygienist's work to quantify the exposure."

Stewart's dose calculation is reliable based on sound methodology, we conclude Butler's causation opinion based on epidemiological studies is likewise no evidence of causation.

As emphasized in *Havner* and *Bostic*, to establish causation through epidemiological studies, Hood needed to show the conditions of the studies were substantially similar to his circumstances. *Havner*, 953 S.W.2d at 720; *Bostic*, 439 S.W.3d at 359. This burden includes proof that (1) he was exposed to the same substance, (2) the exposure or dose levels were comparable or greater than those in the studies, (3) the exposure occurred before the onset of injury, and (4) the timing of the onset of injury was consistent with that experienced by those in the study. *Havner*, 953 S.W.2d at 720. We also must consider the confidence level or interval of the studies to determine if an increased risk of developing AML is statistically significant. *Havner*, 953 S.W.2d at 723–24.

We begin by discussing those studies, referred to as the "painter studies," which Butler relied on, in part, to support her causation opinion: (1) Miller,[16] (2) Lindquist,[17] (3) Lasarov,[18] and (4) Brown.[19] DuPont argues the "painter studies" violate two principles: (1) the "same substance" mandate of *Havner*, and (2) the "essential teaching of *Flores*" and *Bostic* that "dose matters." Our analysis includes Butler's testimony regarding her interpretations of the studies as well as our review of the studies, which are in the record.

1. The Miller Study

The Miller study involved artistic painters. The subjects often worked forty or more hours a week in the studio and ate and slept in inadequately ventilated spaces without appropriate

[16] Barry A. Miller, et al., *Cancer Risk Among Painters*, AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 9:281 (1986).

[17] R. Lindquist, et al., *Increased Risk of Developing Acute Leukemia After Employment as a Painter*, CANCER 60:1378 (1987).

[18] Dusica Lasarov, et al., *Acute Myeloid Leukemia and Exposure to Organic Solvents–A Case-control Study*, EUROPEAN JOURNAL OF EPIDEMIOLOGY 16:295 (2000).

[19] Linda Morris Brown et al., *Exposures in the Painting Trades and Paint Manufacturing Industry and Risk of Cancer Among Men and Women in Sweden*, JOURNAL OF OCCUPATIONAL AND ENVIRONMENTAL MEDICINE vol. 4, No. 3 (2002).

protective equipment. The study found "the leukemia excess among male painters was most pronounced for the myeloid cell type," which Butler testified included AML.[20]

Based on Hood's deposition testimony, Butler believed Hood used substantially similar benzene-containing-type products as those in the study. However, the abstract at the beginning of Miller's study acknowledges "[i]nformation was not available to determine exposure to specific substances that may have been responsible for" the "excess death from leukemia."

In fact, the study stated its intention "was to generate a hypothesis that could be further tested in more analytic studies. . . It was not possible in this study to establish a connection between specific substances used by artists and cancer mortality because such information was not available." The study ended by encouraging future studies to "investigate relationships between specific exposures and chronic health effects."

To the extent the Miller study found a statistically significant increased risk of leukemia in artistic painters, the findings do not support Butler's causation opinion that Hood's exposure to benzene in paint products caused his AML. First, the study neither identified the specific substances used by the painters nor determined the painters' exposures to such substances. Instead, "those selected . . . tend[ed] to be established artists and it is reasonable *to assume* that they worked for several years in their field." [Emphasis added.] However, epidemiological studies are without evidentiary significance if the injured person cannot show his exposure or dose level is comparable to or greater than those in the studies. *Flores*, 232 S.W.3d at 771 (quoting *Havner*, 953 S.W.2d at 720). Without knowing the substances, the dose, and the exposure of those painters in the Miller study, Butler could not compare Hood's lifetime benzene dose to determine whether his exposure was comparable to or greater than those in the study. Further, Hood's work environment was not

[20] The proportionate mortality rate (PMR) equaled 4.0, which showed more than a doubling of the risk of developing leukemia, with a 95% confidence interval of 1.7–9.2, meaning the results are statistically significant.

substantially similar to those painters in the Miller study. Unlike the painters in the Miller study, Hood wore a respirator and worked in a ventilated paint booth—two conditions that impact a worker's exposure to potential carcinogens. As such, the Miller study cannot support Butler's causation opinion.

2. The Lindquist Study

The Lindquist study compared exposure of organic solvents in painters with acute leukemias. The study found a doubling of the risk of professional painters developing acute leukemias with a confidence interval of 1.95 to 554, meaning the results are statistically significant. However, like the Miller study, the Lindquist study does not identify an estimation or average of cumulative benzene exposure. Rather, "the painters in the study reported daily exposure to organic solvents by vapors." The study is silent on any further data regarding the "daily exposure" of those in the study. Further, the painters all reported cleaning their hands with solvents containing a mixture of aromatic and aliphatic hydrocarbons. The study is silent as to what these hydrocarbons were or the amounts used by the painters to clean their hands.

Without knowing the substances, the dose, and the exposure of those painters in the Lindquist study, Butler could not compare Hood's lifetime benzene dose to determine whether his exposure was comparable to or greater than those in the study. Moreover, the record does not indicate Hood cleaned his hands daily with benzene-containing solvents.[21] As such, the Miller study cannot support Butler's causation opinion. *See Flores*, 232 S.W.3d at 771 (quoting *Havner*, 953 S.W.2d at 720); *Bostic*, 439 S.W.3d at 355 ("dose matters").

[21] At most, the record indicates he sometimes got paint or lacquer thinner on his hands while wiping down the trailers and washing his paint gun at the end of each day. He did not wear gloves during either activity.

3. The Lazarov Study

The Lazarov study was a case-control study hypothesizing about the relationship between AML and organic solvents. Exposure to solvents was associated with a significantly increased risk of AML;[22] however, the "solvents are often mixed and we could not determine which specific solvent or solvents each worker was exposed." The study specifically stated, "We made no attempt to categorize the type of solvents to which exposure had occurred." In fact, the study concluded with a cautionary word:

> This study has confirmed an association between AML and solvent exposure but since such is relatively common, and since we were unable to determine any objective measure of exposure the results need to be interpreted with caution. There is clearly a need for further studies in which some measure of "dose" can be determined with more precision.

Butler, however, relied on this study because it involved painters and found that "painters and related workers" exposed to organic solvents had an increased risk of developing AML. Interestingly, there was no statistically increased risk of developing AML from paint.[23]

The study does not define which types of organic solvents the painters were exposed to or the types of jobs performed when painters were exposed. In fact, the study "made no attempt to categorize the type of solvents to which exposure has occurred." Rather, "solvent exposure was assumed to occur in the following operations: painting and surfacing; degreasing metal components; maintenance of materials, machines or the work place; hand cleaning by working using paints or lubricants; and any job entailing the direct handling of solvents or fuels." Butler merely assumed, based on those categories, that some of the Lazarov subjects performed tasks similar to those performed by Hood. When pressed further on whether her answer was based on assumptions, Butler responded, "That's all I can go on, sir. I didn't write the study."

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[22] The odds-ratio is 2.52 with a confidence interval of 1.45–4.39, meaning the results are statistically significant.

[23] The confidence interval is 0.68–2.43. Because it includes 1.0, it is not statistically significant. *See Havner*, 953 S.W.2d at 723.

Again, Butler relied on a study that did not provide the dose exposure of those in the study and did not identify which types of solvents were used within a variety of occupational duties. Without these vital pieces of information, the conditions of this study are not substantially similar to those in this case and cannot establish causation. *See Flores*, 232 S.W.3d at 771 (quoting *Havner*, 953 S.W.2d at 720); *Bostic*, 439 S.W.3d at 355 ("dose matters").

4. The Brown Study

The Brown study considered the increased risk of leukemia, among other cancers, between men and women in Sweden after exposure in the painting manufacturing industry. Among male painters, lacquerers, and pictorial artists, the study failed to show a statistically increased risk of developing leukemia. Only Table 4—The SIRs (standardized incidence ratio) of Cancers Among Male Workers in the Paint and Varnish Industry During 1960 and 1970—showed a slight statistically increased risk of developing nonlymphocytic leukemia with a confidence interval of 1.1–3.6. However, Butler did not know if Hood had ever worked with varnishes. Instead, she justified her reliance on Table 4 because it included painters. When asked, "What was the contribution to these calculations of the exposures to varnish?," Butler answered, "They don't determine that in this study sir, as you know." Yet, despite knowing the study did not provide such information,[24] she opined "the increased risk of cancer was due to the benzene in those products that were used." The study itself, however, is less substantiated and states that "[s]olvents, which *may* include benzene exposure, *likely* account for the increased risk of nonlymphocytic leukemia in our study." [Emphasis added.]

Butler's reliance on this study is further suspect because data in Table 4 relating to acute nonlymphocytic leukemia (ANLL), which is another name for AML, does not show a statistically

[24] In fact, the author acknowledged that a limitation of the study was the inability to evaluate specific chemical exposures through specific job exposures.

significant increased risk for ANLL for paint and varnish workers. The confidence interval is 1.0–4.2. Butler acknowledged this was "technically not statistically significant," but opined the number of painters and varnish workers in the study was small "so medically, it is significant that in such a small number, they still show greater than two increased risk of development of the disease."[25] However, the standard is not whether a study is medically significant but whether it is statistically significant. *Havner*, 953 S.W.2d at 723–24 (if the confidence interval includes 1.0, the study is inconclusive because the confidence interval includes relative risk values that are both less than and greater than the null hypothesis (1.0), leaving the researcher with results suggesting the null hypothesis should be both accepted and rejected). An expert cannot dissect a study, picking and choosing data to derive a higher relative risk if the process does not comport with sound scientific methodology. *Id*. at 720; *Mo. Pac. R. Co. v. Navarro*, 90 S.W.3d 747, 754 (Tex. App.—San Antonio 2002, no pet.) (reversing trial court judgment based on unreliable expert's testimony regarding epidemiological studies).

Similar to the other "painter studies," the Brown study suffers from the same flaws. Without knowing which solvents were used and how much the painters were exposed, the study is not substantially similar to the facts in Hood's case. *See Flores*, 232 S.W.3d at 771 (quoting *Havner*, 953 S.W.2d at 720); *Bostic*, 439 S.W.3d at 355. Further, the Brown study did not find a statistically significant increased risk of a painter or varnish worker developing AML. *See, e.g., Matt Dietz Co. v. Torres*, 198 S.W.3d 798, 804 (Tex. App.—San Antonio 2006, pet. denied) (expert testimony did not support jury verdict when, among other things, "none of the studies identify a specific substance or a group of substances as having a statistically significant

[25] We note that during this testimony, Butler refers to "nonlymphocytic leukemia"; however, given her reference to a confidence interval that includes 1.0 and our review of Table 4, Butler is referring to acute nonlymphocytic leukemia.

association with laryngeal cancer"). Accordingly, Butler's reliance on the Brown study to support her opinion is no evidence of causation.

After reviewing Butler's testimony, along with the "painter studies," we conclude such evidence is unreliable and therefore amounts to no evidence to support the jury's verdict that the benzene in DuPont's products caused Hood to develop AML. However, Butler testified to additional studies involving benzene exposures from crude oil and petroleum: (1) Guénel,[26] (2) Glass,[27] (3) Shnatter,[28] (4) Kirkeleit,[29] and (5) Vlaanderen.[30] None of these studies involved painters in working conditions similar to the conditions where Hood worked. This alone undermines Butler's reliance on them. Regardless, Hood argues the studies establish a statistically significant relationship between benzene and AML and even if these studies involve liquid mixtures that are not the same as paint, the mixtures contained benzene—essentially arguing benzene is benzene regardless of what comprises the rest of the mixture. We consider each study in turn.

5. **The Guénel Study**

The Guénel study investigated the risk of leukemia in a large cohort of gas and electric utility workers with exposures to several suspected or confirmed carcinogens. The results showed an increased risk of 3.6 over the unexposed population, meaning the exposed population was three and a half times more likely to develop leukemia as a result of exposure.[31] The study also reached

[26] Pascal Guénel, et al., *Leukemia in Relation to Occupational Exposures to Benzene and Other Agents: A Case-Control Study Nested in a Cohort of Gas and Electric Utility Workers*, AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 42:87 (2002).

[27] Deborah C. Glass, et al., *Leukemia Risk Associated With Low-Level Benzene Exposure*, EPIDEMIOLOGY vol. 14, no. 5 (Sept. 2003).

[28] A. Robert Schnatter, et al., *Myelodysplastic Syndrome and Benzene Exposure Among Petroleum Workers: An International Pooled Analysis*, JOURNAL OF THE NATIONAL CANCER INSTITUTE (2012).

[29] Jorunn Kirkeleit, et al., *Increased Risk of Acute Myelogenous Leukemia and Multiple Myeloma in a Historical Cohort of Upstream Petroleum Workers Exposed to Crude Oil*, CANCER CAUSES CONTROL 19:13 (2008).

[30] Jelle Vlaanderen, et al., *Occupational Benzene Exposure and the Risk of Lymphoma Subtypes: A Meta-Analysis of Cohort Studies Incorporating Three Study Quality Dimensions*, ENVIRONMENTAL HEALTH PERSPECTIVES vol. 119, no. 2 (Feb. 2011).

[31] The confidence interval is 1.1–11.7.

statistical significance for benzene exposure and all acute leukemias. The study, however, did not reach a level of statistical significance for benzene exposure and AML specifically.

The study focused on an "unadjusted odds ratio," which indicated an increased risk of leukemia with cumulative exposure to benzene. However, once the odds ratio was adjusted, benzene exposure arising from the use of solvents was no longer statistically significant.[32] Butler admitted she was "not clear, based on the way they describe the study, exactly what they did to adjust." And while ratio of benzene exposure to all acute leukemias showed statistical significance[33] based on the unadjusted odds ratio, the study showed no statistical significance for AML.[34] We cannot treat an epidemiological study regarding one disease as if it applies to another. *See, e.g., Exxon Corp. v. Makofski*, 116 S.W.3d 176, 186 (Tex. App.—Houston [14th Dist.] 2003, pet. denied) ("While lumping distinct diseases together as 'leukemia' may yield a statistical increase as to the whole category, it does so only by ignoring proof that some types of the disease have a much greater association with benzene than others.").

The study noted that although the results were no longer statistically significant when only the use of solvents was taken into consideration, the risk only slightly decreased. Hood claims this "confirmed" that "the benzene exposure from solvents contributes *by its own* to the excess risk of leukemia." Hood has omitted an important word from Guénel: "indicate." The study did not confirm but "indicate[d]" benzene exposure from solvents contributed on its own to excess risk of leukemia.

The study also acknowledged several limitations: (1) exposure miscalculations for different reasons such as factory workers located in different parts of the country and (2) "assessment of

[32] The unadjusted odds ratio is 3.6 with a confidence interval of 1.1–11.7. The adjusted odds ratio is 3.1 with a confidence interval of 0.3–30.4.

[33] The odds ratio is 4.6 with a confidence interval of 1.2–17.4.

[34] The odds ratio is 2.4 with a confidence interval of 0.7–8.5.

exposure to chemicals was *based on expert judgment*, since *no exposure monitoring data was available*." (emphasis added) "Because of uncertainties regarding the true exposure levels, we used arbitrary units of benzene concentration to estimate exposure intensity." Further, Guénel noted the following:

> Although benzene exposure is thought to be linked with AML [Wong, 1995], it has also been shown in a literature review that an exclusive relationship with this leukemia subtype is based on insufficient evidence. . . .
>
> In the present study, the increased risk of leukemia is primarily associated with acute leukemia subtypes. It is suggested that both acute myeloid and acute lymphoid leukemias are at increased risk. However, the analysis is limited due to small numbers.

Guénel concluded that although benzene was "more clearly associated with acute leukemia than with chronic leukemia, no clear association with a specific subtype appear from our data," meaning the study did not support a finding that benzene exposure caused AML. In fact, Guénel believed "further follow-up of these workers . . . may help to clarify the results."

Given the limitations of the study regarding exposure data, the lack of statistical significance between benzene and AML specifically, and Butler's inability to explain the adjustments used in the study, her reliance on this study to support her opinion is no evidence of causation. *See Navarro,* 90 S.W.3d at 754 ("an expert cannot dissect a study, picking and choosing data . . . to derive a higher relative risk"); *see also Havner*, 953 S.W.2d at 723–24 (to be statistically significant, confident interval cannot include 1.0); *Bostic*, 439 S.W.3d at 347 (study must show injured person was exposed to same substance and exposure levels were comparable to or greater than those in the study).

6. The Glass Study

Next, Butler discussed the Glass study, which found that people exposed to greater than 16 ppm years had 98 times over the unexposed population risk of developing leukemia from benzene

exposure.[35] The results included all leukemias, which thereby included AML. It involved five out of twenty-two petroleum workers who were exposed to products with 70% benzene, not 0.1–0.5% benzene (according to Stewart) as in DuPont's products. Butler testified the difference in occupation of those in the study did not matter because "whether it's a mixture of paint, or a part of crude oil, it's still a mixture. The body is metabolizing the benzene portion of whatever is coming in." But, Butler could not say a benzene-containing solvent in the petroleum industry and a benzene-containing paint thinner in the painting industry with the same percentages of benzene and with the same cumulative dose would translate to the same cancer risk.

Despite *Havner*'s admonition that epidemiological studies must be substantially similar and involve the same substance, she testified "even though there is not a study specifically about painters, [Glass] is a study that looks at people exposed to benzene in a mixture as part of something else." However, her opinion overlooks the fact courts and federal agencies have found gasoline, a mixture containing benzene, does not cause AML. *See, e.g., Castellow*, 97 F. Supp. 2d at 798 ("The pertinent studies show that persons exposed to gasoline, by occupation, do not reflect a statistically significant excess rate of AML."); *see also Burst v. Shell Oil Co.*, 120 F. Supp. 3d 547, 553 (E.D. La. 2015) ("the simple explanation that gasoline contains benzene, and benzene is a known carcinogen cannot be the justification for such extrapolation" particularly when multiple agencies concluded that although benzene is a carcinogen, it has not reached the same conclusion regarding gasoline), *aff'd*, 650 Fed. Appx. 170, 173 (5th Cir. May 23, 2016). She cites to no study in which painters exposed to benzene from solvents in paint have a statistically significant increased rate of AML.

The study acknowledged its limitations, which included its relatively small number of cases (thirty-three leukemias of which there were only eleven acute nonlymphatic leukemias and

[35] The odds ratio is 98.2 with a confidence interval of 8.8-1090.

specifically nine AMLs). Such small numbers limited its power to detect excess risks for leukemia subgroups, "particularly when we stratified the subjects by exposure." We cannot treat an epidemiological study regarding one disease as if it applies to another. *See, e.g, Makofski*, 116 at 186 ("While lumping distinct diseases together as 'leukemia' may yield a statistical increase as to whole category, it does so only by ignoring proof that some types of the disease have a much greater association with benzene than others.").

We acknowledge that Table 6, Association of Leukemia Subtype With Cumulative Benzene Exposure From Conditional Logistic Regression Analysis, provides the following:

Cumulative Lifetime Benzene Exposure (ppm-years)	ANLL (N=11)
>4-8	0.52 (0.05-5.0)
>8	7.17 (1.27-40.4)

This shows a risk assessment for developing ANLL of 7.17 for those petroleum workers with benzene exposure estimates of greater than 8ppm-years—meaning, a person exposed to a level of greater than 8 ppm-years had a seven-fold increased risk of developing ANLL. Hood argues because his exposure, which Stewart approximated at 29.98 ppm years, was well in excess of 8 ppm-years as found in this study, the Glass study supports Butler's opinion. Given our previous discussion of Stewart's methodology for calculating Hood's lifetime benzene exposure, we are not persuaded by this argument. Further, the lowest end of Stewart's calculation for Hood's lifetime benzene exposure was 8 ppm-years. The Glass study did not find a statistically significant increased risk of developing ANLL for those in the cohort with a lifetime benzene exposure between 4–8 ppm-years. Accordingly, even putting aside the fact the study involved different workers in a different industry, the Glass study could not support Butler's opinion based on even

Stewart's lowest average calculation of Hood's lifetime benzene exposure dose. As such, Butler's reliance on this study to support her opinion is no evidence of causation.

7. **The Schnatter Study**

The Schnatter study is a pooled analysis study examining the risk of developing five lymphohematopoietic cancers from relatively lower levels of benzene exposure in petroleum workers. The study focused on terminal workers and tanker drivers. The author "updated three nested-case control studies from Australia, Canada, and the United Kingdom." The Australian study was the previously discussed Glass study. To the extent Glass found a statistically significant increased risk of developing ANLL from benzene, the results were not replicated in the Schnatter study.[36] This further undermines Butler's reliance on Glass. *See, e.g., Daniels v. Lyondell-Citgo Ref. Co.*, 99 S.W.3d 722, 729–30 (Tex. App.—Houston [1st Dist.] 2003, no pet.) (concluding that reliance on epidemiological studies with later updates that failed to establish a doubling of the risk for cancer could not support general causation to survive summary judgment); *see also Havner*, 953 S.W.2d at 727 ("[I]t is important that any conclusions about causation be reached only after an association is observed in studies among different groups and that the association continues to hold when the effects of other variables are taken into account.").

The Schnatter study found an association with an increased risk of developing myelodysplactic syndrome (MDS, considered a preleukemic process) at relatively low-level exposures to benzene in petroleum distribution workers "but not AML." Butler found this study supportive because Hood's records indicated he was treated for MDS, which was more likely to progress to AML.[37] However, Hood's theory of the case was that benzene from DuPont's paint

[36] The study stated: "Indeed, the original Australian study (13) reported a strong relationship with AML, but this was not replicated in this pooled data, partly because of the absence of a relationship for updated case subjects and partly because of reclassification of some AML case subjects to MDS."

[37] Butler never saw medical records with Hood's alleged original MDS diagnosis; however, she believed he "must have" presented with it based on his course of treatment for months preceding his AML diagnosis.

products caused him to develop AML. Butler was specifically designated to testify that "to a reasonable degree of medical certainty Mr. Hood's AML was caused by his history of occupational exposures to benzene from products manufactured, supplied, or sold by" DuPont. Her ultimate opinion at trial was that DuPont's benzene-containing paint products more likely than not contributed to Hood's AML.

Butler cherry-picked data from one table within this study showing a statistically significant increased risk of developing MDS for cumulative exposure. The same table did not show a statistically significant increased risk of developing AML for cumulative exposure. Moreover, the data did not show a statistically significant increased risk of workers developing AML or MDS at average or maximum exposure intensity. To rely on a study that affirmatively shows the opposite of her causation opinion—meaning no statistically significant increased risk of AML from benzene exposure—is not scientifically reliable evidence to support a verdict. *See Havner*, 953 S.W.2d at 730 (expert's testimony based on epidemiological studies that concluded "just the opposite" was not reliable evidence to support jury verdict). Butler did not reconcile her reliance on a single statistic despite the other contrary findings. An expert's assurance that a study establishes causation does not make it so. *See In re Allied Chem. Corp.*, 227 S.W.3d 652, 656 (Tex. 2007). Accordingly, Butler's reliance on this study to support her opinion is no evidence that benzene caused Hood's AML.

8. The Kirkeleit Study

This study included upstream petroleum workers exposed to crude oil and found a statistically significant increased risk of developing AML with an odds ratio of 3.67 and a confidence interval of 1.3 to 10.34. Butler did not provide any further information about the study except this conclusory statement that it supported a causal link between benzene and AML. She did, however, admit crude oil contains different materials, some of which are carcinogenic.

Our review of the study indicates workers in the study were exposed to benzene through contact with crude oil and natural gas, none of which were substances similar to Hood's exposure. A "major limitation of [the] study" was lack of exposure estimates, which is critical. Butler conceded the study did not include any specific quantitative measurements that estimated the workers' cumulative or average exposure.[38] Without such information, this study is without evidentiary significance. *See Flores*, 232 S.W.3d at 771 (epidemiological studies are without evidentiary support if the injured person cannot show that "the exposure or dose levels were comparable to or greater than those in the study").

9. The Vlaanderan Study

This study was a meta-analysis, meaning the authors used other published studies related to benzene exposure and the development of various lymphomas and AML to prove or disapprove a hypothesis: benzene exposure and risk of leukemia.[39] Butler merely testified, "this was a study that looked at lymphoma subtypes, but they described AML as the standard, because it is accepted that benzene exposure contributed to increased risk for development of AML."

Hood argues the meta-analysis supports causation because it found an odds ratio of 2.32 with a 95% confident interval of 1.55–3.47 for AML based on quantitative exposures assessment "like Mr. Hood's" in Table 4. However, our review of the meta-analysis does not reveal any information about the underlying quantities of benzene to which the subjects in those studies were exposed for a comparison to Hood's benzene exposure. To the extent Hood argues Butler provided a "lengthy explanation" during her pre-trial deposition for her decision to rely upon this study, her explanation included an admission that the meta-analysis did not contain a table analyzing quantitative benzene exposure and she did not know which nine studies supported the data in Table

[38] In a supplemental expert report filed June 12, 2015, Butler stated "the benzene exposure estimate was not quantitated in this study."

[39] The meta-analysis included Glass, Guénel, and Kirkeleit.

4.[40] Therefore, reliance on the meta-analysis is "problematic" without dose information. *See, e.g., Merck & Co. v. Garza*, 347 S.W.3d 256, 267 (Tex. 2011) (reliance on meta-analysis was "especially problematic" when it combined results from a number of different studies, with differing drug dosages, durations, and comparison drugs); *Bostic*, 439 S.W.3d at 347 (study must show injured person was exposed to same substance and exposure levels were comparable to or greater than those in the study). Essentially, Butler provided nothing more than a conclusory statement that the meta-analysis established causation between benzene and AML. An expert's assurance that a study establishes causation does not make it so. *See In re Allied Chem. Corp.*, 227 S.W.3d at 656.

10. Conclusion

After thorough and careful review of Hood's contentions, as well as the record testimony, exhibits, and epidemiological studies, we conclude Hood failed to demonstrate the reliability of his scientific evidence because the studies relied on by Butler either failed to be substantially similar to his circumstances or they failed to quantify the benzene exposure thereby providing a dose to which Butler could compare (assuming the reliability of Stewart's dose calculation). At most, he provided one study, the Glass study, in support of his argument that his dose of benzene exposure caused him to develop AML; however, this conclusion is only supported by assuming (which we do not) that Stewart's lifetime benzene exposure dose calculation is correct. Because her testimony is unreliable, it is no evidence of causation and cannot survive a sufficiency challenge. *See Havner*, 953 S.W.2d at 713; *see also Bostic*, 439 S.W.3d at 359.[41] We sustain DuPont's first issue.

[40] She testified in her deposition, "I couldn't answer that without really going into detail." When pressed on whether she knew the quantitative measurements in any or all of the nine studies that were characterized, she answered she "would have to go back to each study."

[41] DuPont also argues Butler's studies failed to show that the timing of Hood's onset of AML was consistent with the latency period of others. However, we need not address latency in light of our conclusions regarding the reliability of other epidemiological studies. TEX. R. APP. P. 47.1.

Having sustained DuPont's first and second issues, we need not address its remaining arguments. *See* TEX. R. APP. P. 47.1. In light of our disposition, we need not address Hood's cross-issues. *See id*.

Conclusion

The evidence of causation is legally insufficient to sustain the verdict in this case. We reverse the judgment of the trial court and render a take-nothing judgment in favor of DuPont.

/David L. Bridges/
DAVID L. BRIDGES
JUSTICE

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Court of Appeals
Fifth District of Texas at Dallas

JUDGMENT

E.I. DU PONT DE NEMOURS AND COMPANY, Appellant

No. 05-16-00609-CV V.

VIRGIL HOOD AND LORRIE HOOD, Appellees

On Appeal from the 160th Judicial District Court, Dallas County, Texas
Trial Court Cause No. DC-13-03619.
Opinion delivered by Justice Bridges.
Justices Myers and Schenck participating.

In accordance with this Court's opinion of this date, the judgment of the trial court is **REVERSED** and judgment is **RENDERED** that appellees Virgil Hood and Lorrie Hood take nothing on their claims against appellant E.I. Du Pont de Nemours and Co.

It is **ORDERED** that each party bear their own costs of this appeal.

Judgment entered May 8, 2018.